DISSERTATION ON

RELATION BETWEEN COPD, SMOKING AND DEPRESSION AMONG ELDERLY POPULATION

SUBMITTED TO THE TAMIL NADU DR.M.G.R. MEDICAL UNIVERSITY
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DECLARATION

I solemnly declare that the dissertation entitled "**Relation Between COPD, Smoking And Depression Among Elderly Population**" is done by me at Madras Medical College and Govt. General Hospital, during 2004-2007 under the guidance and supervision of Prof.B.Krishnasamy, M.D. This dissertation is submitted to the Tamil Nadu Dr.M.G.R. Medical University towards the partial fulfillment of requirements for the award of M.D. Degree in Geriatric Medicine (Branch - XVI).

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CERTIFICATE

This is to certify that this Dissertation entitled "**Relation Between COPD, Smoking And Depression Among Elderly Population**" is a bonafide work done by Dr.SENTHIL RAJA PERUMAL.L M.D. (Geriatric Medicine) postgraduate of Department Of Geriatric Medicine, Madras Medical College, Chennai, during the academic year 2004-2007.

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CONTENTS

Introduction

INTRODUCTION

The remarkable success of medicine and public health over the past century has made it possible for elderly people to live longer than ever before. At present, people over 65 years account for 13% of World population, which will swell to about 20% by 2030, as the 'baby boomers' age. Thus most physicians will spend a significant portion of their professional life dealing with health care of older adults (1).

As per WHO and National Policy for Older Persons, the age used to define Geriatric persons in India is 60 years. Life expectancy in India is 62.8 years in males and 64 years in females. (2) In India, the expectation of life at birth has increased from about 33 years in 1941-51 to over 60 years in 1991-2001. During past 20 years the life expectancy has increased roughly half a year per annum (3).

The elderly population in India is the second largest in the world, next only to China. This population which was 77 million according to 2001 census (7.5% of total population) is projected to increase to 137 million in 2021. Their annual growth is higher (3%) when compared to growth of entire population (1.9%). Trends show that by 2050, the elderly population of India will surpass the population of children below

14 years. Thus in India also, the elderly population is on an increase, as in other regions of the globe. This population is more likely to present with chronic diseases and atypical manifestations of diseases. COPD is one of the chronic diseases found in this population.

COPD is currently the fourth leading cause of death (4, 5, 8). COPD is a major cause of disability and death in most countries and contribute significantly to escalating health care costs (6). In a recent estimate of Disability Adjusted Life Years (DALYs) lost due to a chronic disease, COPD ranked ninth in the world (7). It is predicted that in 2020, COPD will be the third leading cause of death in the world and fifth leading cause of lost DALYs worldwide(8). So, it is necessary to concentrate more on a disease which is not only prevalent but also increasing in prevalence year by year.

COPD is caused by smoking in 80-90% of cases(6). The only known measure found to decrease the incidence of COPD and decrease its severity is smoking cessation (9).

Tobacco is responsible for half of all cancers in men and 25% of all cancers amongst women (10). 15% of smokers develop COPD.

Quitting smoking by 50% of adults would reduce the mortality amongst smokers by tobacco to 33% by 2020(11).

It has been found that smoking is related to depression (12, 13). Studies show that genes for smoking and depression are closely related and hence cause doubling of depression amongst smokers.

Depression affects 10-12% of elderly attending primary care as per Current Geriatrics Diagnosis and Treatment, 2004 edition. The incidence of depression is much higher in Indian population compared to western data.

The occurrence of depression in COPD patients increases the morbidity and mortality in the latter. Indeed studies have shown that depression plays a larger role in determining a patient's quality of life than the severity of COPD(4). More alarming is the fact that <30% of health care providers recognize depression. Depression compounds the negative symptoms of COPD leading to longer hospital stays and higher mortality rates. So, diagnosis and treatment of depression is a must in all COPD patients (4).

Smoking is associated with both depression (12, 13) and COPD. Smoking, more than adds to the amount of depression in COPD patients.

Paradoxically, studies show that nicotine has anti-depressant effect in short term but adds to depression in long term. People who quit smoking are more prone to depression (14) over the next 6 months and so, they cannot leave smoking. So, a vicious cycle results.

So, this study was undertaken, to find out whether smoking per se is related to depression and if so whether it adds to depression in people with COPD.Since patients with COPD are nearly always smokers this study also tends to find out whether they are much more depressed than non-smoking controls. This is to stress that all COPD patients require screening for depression and if present should be appropriately treated.

Review of Literature

REVIEW OF LITERATURE

CHRONIC OBSTRUCTIVE PULMONARY DISEASE

Definition

COPD is a disease state characterized by airflow limitation that is not fully reversible. The airflow limitation is usually both progressive and associated with an abnormal inflammatory response of the lungs to noxious particles or gases.(15)

A diagnosis of COPD should be considered in any patient who has symptoms of cough, sputum production, or dyspnea, and/or a history of exposure to risk factors for the disease. The diagnosis is confirmed by spirometry. The presence of a post bronchodilator FEV_1/FVC <70% confirms the presence of airflow limitation that is not fully reversible. Where spirometry is unavailable, the diagnosis of COPD should be made using all available tools. Clinical symptoms and signs, such as abnormal shortness of breath and increased forced expiratory time, can be used to make the diagnosis. A low peak flow is consistent With COPD, but has poor specificity since it can be caused by other lung diseases and by poor performance. In the interest of improving the

diagnosis of COPD, every effort should be made to provide access to standardized spirometry. Chronic cough and sputum production often precede the development of airflow limitation by many years; but not all individuals with cough and sputum production go on to develop COPD (15).

Classification of Severity

For educational reasons, a simple classification of disease severity into four stages is recommended. The management of COPD is largely symptom driven, and there is only an imperfect relationship between the degree of airflow limitation and the presence of symptoms. The staging, therefore, is a pragmatic approach aimed at practical implementation and should only be regarded as an educational tool, and a very general indication of the approach to management. All FEV_1 values refer to post bronchodilator FEV_1.

Stage 0 : At Risk - Characterized by chronic cough and sputum production. Lung function, as measured by spirometry, is still normal.

Stage I : Mild COPD- Characterized by mild airflow limitation (FEV_1 /FVC_1 < 70% but $FEV_1 \geq$ 80%

predicted) and usually, but not always, by chronic cough and sputum production. At this stage, the individual may not even be aware that his or her lung function is abnormal.

Stage II : Moderate COPD - Characterized by worsening airflow limitation (50% ≤ FEV1 <80% predicted) and usually the progression of symptoms, with shortness of breath typically developing on exertion. This is the stage at which patients typically seek medical attention because of dyspnea or an exacerbation of their disease.

Stage III : Severe COPD - characterized by further worsening of airflow limitation (30% ≤ FEV_1 <50% predicted), increased shortness of breath, and repeated exacerbations which have an impact on patients' quality of life.

Stage IV : Very Severe COPD - Characterized by severe airflow limitation (FEV_1 <30% predicted). At this

stage, quality of life is appreciably impaired and exacerbations may be life-threatening (15).

Poorly reversible airflow limitation associated with bronchiectasis, cystic fibrosis, tuberculosis, or asthma is not included except in so far as these conditions overlap with COPD. In many developing countries both pulmonary tuberculosis and COPD are common. Therefore, in all subjects with symptoms of COPD, a possible diagnosis of tuberculosis should be considered especially in areas where this disease is known to be prevalent. In countries in which the prevalence of tuberculosis is greatly diminished, the possible diagnosis of this disease is sometimes overlooked.

Pathogenesis

COPD is characterized by chronic inflammation throughout the airways, parenchyma, and pulmonary vasculature. Macrophages, T lymphocytes (predominately $CD8^+$), and neutrophils are increased in various parts of the lung (15).

In addition to inflammation, two other processes thought to be important in the pathogenesis of COPD are an imbalance of proteases and anti-proteases in the lung, and oxidative stress.

Inflammation of the lungs is caused by exposure to inhaled noxious particles and gases. Cigarette smoke can induce inflammation and directly damage the lungs.

Pathology

Pathological changes characteristic of COPD are found in the central airways, peripheral airways, lung parenchyma, and pulmonary vasculature.

In the central airways -the trachea, bronchi, and bronchioles greater than 2-4 mm in internal diameter - inflammatory cells infiltrate the surface epithelium. Enlarged mucus secreting glands and an increase in the number of goblet cells are associated with mucus hyper secretion. In the peripheral airways -small bronchi and bronchioles that have an internal diameter of less than 2 mm -chronic inflammation leads to repeated cycles of injury and repair of the airway wall. The repair process results in a structural remodeling of the airway wall, with increasing collagen content and scar tissue formation that narrows the lumen and produces fixed airways obstruction.

Destruction of the lung parenchyma in COPD patients typically occurs as centrilobular emphysema. This involves dilatation and

destruction of the respiratory bronchioles. These lesions occur more frequently in the upper lung regions in milder cases, but in advanced disease they may appear diffusely throughout the entire lung and also involve destruction of the pulmonary capillary bed (15).

Pathophysiology

Pathological changes in the lungs lead to corresponding physiological changes characteristic of the disease, including mucus hyper secretion, ciliary dysfunction, airflow limitation, pulmonary hyperinflation, gas exchange abnormalities, pulmonary hypertension, and cor pulmonale. They usually develop in this order over the course of the disease.

Mucus hyper secretion and ciliary dysfunction lead to chronic cough and sputum production. These symptoms can be present for many years before other symptoms or physiological abnormalities develop.

Expiratory airflow limitation, best measured by spirometry, is the hallmark physiological change of COPD and the key to the diagnosis of the disease. It is primarily due to fixed airways obstruction and the consequent increase in airways resistance. Destruction of alveolar

attachments, which inhibits the ability of the small airways to maintain patency, plays a smaller role (15).

Epidemiology

In the Global Burden of Disease Study conducted under the auspices of the WHO and the World Bank, the worldwide prevalence of COPD in 1990 was estimated to be 9.34/1,000 in men and 7.33/1,000 in women.

The limited data that are available indicate that morbidity due to COPD increases with age and is greater in men than women.

COPD is currently the fourth leading cause of death in the world, and further increases in the prevalence and mortality of the disease can be predicted in the coming decades (15).

Risk factors

Risk factors for COPD include both host factors and environmental exposures, and the disease usually arises from an interaction between these two types of factors. The host factor that is best documented is a rare hereditary deficiency of alpha-1 antitrypsin.

Other genes involved in the pathogenesis of COPD have not yet been identified. The major environmental factors are tobacco smoke; heavy exposure to occupational dusts and chemicals (vapors, irritants, fumes); and indoor/outdoor air pollution (15).

The ATS Definition of COPD

Internationally accepted opinion, including the 1995 ATS statement, has defined COPD as a disease state characterized by chronic airflow limitation due to chronic bronchitis and emphysema. Chronic bronchitis has been defined in clinical terms: the presence of chronic productive cough for at least 3 consecutive months in 2 consecutive years. Other causes of chronic productive cough must be ruled out. Emphysema, on the other hand, has been defined by its pathologic description: an abnormal enlargement of the air spaces distal to the terminal bronchioles accompanied by destruction of their walls and without obvious fibrosis. The new ATS/ERS guidelines, like the GOLD guidelines, have parted from this traditional description of COPD, Similar to the changes in the definition of asthma by the NHLBI more than a decade ago, the definition of COPD has undergone major revision. COPD, like asthma, is now recognized as an inflammatory disease of the airways. This is supported by extensive clinical and basic

science research over the past 2 decades showing that asthma and COPD have different and distinct cellular and inflammatory mediator profiles. The new ATS/ERS definition reflects these scientific advances: “Chronic obstructive pulmonary disease (COPD) is a preventable and treatable disease characterized by airflow limitation that is not fully reversible. The airflow limitation is usually progressive and is associated with an abnormal inflammatory response of the lungs to noxious particles or gases, primarily caused by cigarette smoking. Although COPD affects the lungs, it also produces significant systemic consequences (9).

While the new guidelines do not specifically include chronic bronchitis and emphysema in the definition of COPD, it is made clear that they are considered the predominant causes of COPD. Asthma is a different clinical entity from COPD. The airflow obstruction is predominantly reversible, and the airways of asthmatics are markedly hyper responsive to a vast number of specific (aeroallergens) and nonspecific (methacholine, histamine, cold air) inhaled substances.

ERS (European Respiratory Society) defined COPD as “reduced maximum expiratory flow and slow forced emptying of lungs, which is

slow, progressive and mostly irreversible to present medical treatment (16)".

Since the GOLD definition is based on consensus between WHO & NHLBI which is internationally accepted, in this study the GOLD criteria was used to select cases of COPD based on spirometry.

As smokers form 90% of those with COPD, in this study COPD patients only with history of smoking are taken as it is easier to find COPD in smokers rather than in non-smokers. Since they form the bulk of cases in community, they represent the COPD cases in community. Since in this region females rarely smoke, all cases in this study are males with COPD with habit of smoking. Asthma patients have been excluded by reversibility of obstruction with bronchodilator (15).

SMOKING AND COPD

Smokers have higher prevalence of COPD, greater annual decline of $FEV_{1,}$ higher death rates from COPD compared to non-smokers. Smoking cessation is the single most effective and cost effective way to reduce development of COPD and stop its progression (15).

The reduction in FEV_1 per year, above normal decline in adults for each pack year of smoking is 9 ml in males and 6 ml in females (17). However many people with a significant number of pack years can still have a normal FEV_1 (17).

However smoking per se, does not cause COPD. **Augesti et al** in the journal Thorax state that, not all smokers develop COPD (18).

In susceptible smokers, there is an increased inflammatory response in lungs whereas in others there is only mild inflammation caused by smoking. So, some hypothesize that COPD is an autoimmune disease triggered by smoking (18). ATS - Patient's section says that "what determines which patients are susceptible to COPD is not known (19)". A text on airway mucus states that genetic factors may determine individual susceptibility (20). It has been calculated that 15% of smokers will end up with symptomatic COPD (17) and a little more with asymptomatic COPD.

SMOKING

Tobacco is a global problem with 1.1 billion tobacco consumers in the world today. WHO estimates if current trends continue, then by 2025, the number of smokers will increase to 1.64 billion, with most

increase occurring in developing countries. Four million people die yearly from tobacco related diseases, one death in every 8 seconds. India is the third largest producer of tobacco in world next only to China & USA (10).

In India only 20% of total tobacco consumed is as cigarettes. 40% is consumed as bidis with the rest as chewing tobaccos, pan masala, snuff hookah, Hookli and Chumma Dhumti. It is estimated that 65% of all men use some form of tobacco (35% smoking, 22% smokeless tobacco, 8% both). Prevalence among women varies widely (15% in Bhaunagar to 67% in AndraPradesh). However, only 3% of the women smoke. Use of smokeless tobacco by women is equal to that of men (10).

The smoke from bidis contains thrice the amount of nicotine and carbon monoxide and 5 times as much tar as smoke from regular filtered cigarettes. Due to high Nicotine, bidis are more addictive than normal cigarettes (21).

Smoking can be variously defined. One definition is continuous use of bidis, cigarettes or any form of tobacco for more than 6 months (22). Non smokers are those who have never smoked (22).

Smoking was divided as per **criteria of Rastogi et al. in a study in IJMR** as (22)

i) Mild smokers = 1-10 cigarettes or 1-15 bidis per day.

ii) Moderate smokers = 11-20 cigarettes or 16-30 bidis per day.

iii) Heavy smokers = ≥ 21 cigarettes or ≥ 31 bidis per day.

As most of the patients who attend OPD in Government General Hospital attached to Madras Medical College smoke only bidis, for comparison, only patients who smoke bidis were taken as `Smokers' in this study. They should have smoked for at least 6 months and all people labeled as smokers were picked up from Rastogi's moderate smoker class for ease for comparison.

People, who have never taken any tobacco products, smoking or smokeless, were labeled as Non-smokers.

CDC definition of smokers: (23)

Life time smokers = Ever smoked even if just one puff

Current user = Smoked in last 30 days

Frequent user	=	Smoked 20 or more cigarettes in last 30 days
Established user	=	Smoked more than 100 cigarettes in their life time.

All smokers in this study belong to `current users' and `established users' as per CDC definition.

None of the controls used in this study had smoked even a puff.

DEPRESSION

The WHO has ranked depression as the fourth in a list of most urgent health problems world wide (24). Depressive disorders afflict one out of five women and one out of ten men at sometime during their lives (24). Despite availability of effective treatments, many patients with mood disorders are disabled and the rate of suicide is high in young and especially elder men (24). Although depression is more common in women, more men rather than women die because of depression causing suicide (24). Nearly half of all cases of depression go undetected. Because mood disorders underlie 50-70% of all suicides, effective treatment at national level should drastically reduce suicides. That

elderly depressed people, often with medical co-morbidity, constitute the highest risk group for suicide, yet escape clinical detection and treatment is particularly problematic for public health.

Depression can be explained by various models (29) including – 1- Aggression turned inward model of Freud and Karl Abraham 2- Object loss model of Freud 3- Loss of self esteem and helplessness in attaining goals of ego ideal model of Edwin Bribing. 4- Cognitive model with thinking on negative lines model of Aaron Beck 5- Learned helplessness model of Martin 6- Deregulated nor-adrenergic and serotonin mediated transmission 7- Neuronal hyper excitability and 8- Final common pathway which place stress -diathesis interaction finally converging on midbrain.

In the study **In Depression in older people: a point to remember in all specialties, Acharya.A** states that, `Depression is a common disorder in older people. It is usually undiagnosed in elder patients due to atypical symptoms, masked depressive state, mixed with agitation, psychotic delusions and worsening of physical symptoms already present or multiple pains in extremities. It is commonly associated co morbidity in patients of all disciplines--as in post CVA state or post myocardial infarction, postoperative state, post

hysterectomy or in a metabolic medical disorder like diabetes mellitus. **Acharya.A** has studied and analyzed 120 patients in different wards of Midnapore Medical College & Hospital and has found high incidence of depression and a marked improvement of symptoms after a short period of treatment.'(25).However Kaplan and Sadock state that, Depression is less common in old age compared to younger ones. A compromise is achieved by Hazzard Text book of Geriatric medicine whose authors state that Major Depressive Disorder is less common in old age but depressive symptoms are much more common in old age compared to younger people. (26)

The American Psychiatric Association states that, `Major depression and dysthymia may be indicated by presence of depressed mood, loss of interest or pleasure along with loss or gain of weight, sleep disturbance, loss of energy, guilt, poor concentration and thoughts of death (27)'.

Screening for depression can be done by PRIME-MD Scale (as indicated by **Spitzer et al** in their study) (27), Beck depression inventory -II, Hospital Anxiety and depression scale. Zung self rating depression scale (29), Centre for epidemiological studies depression questionnaire (30) and the Geriatric Depression Scale (31)(32).

The Geriatric depression scale is widely used and validated in many languages. It is a 30-item scale. Its 15 item shortened version is often used for ease of administration. Sheikh & Yesavage had recommended use of GDS in Gerontology journal as early as in 1986 (33) (34). This short version is considered positive if score is 5 or more out of 15. **Ada C Mui et al** in their study have found that GDS is valid in Indian Population (35). So, in this study, GDS-15 has been used to screen for depression.

SMOKING AND DEPRESSION

Scientists are still searching for a clear understanding of the link between depression and smoking. One explanation is 'Self-medication Idea', that Nicotine has a short term benefit effect on Neuro-transmitter systems involved in depression, so smoking cigarette may provide some relief from the feelings of depression. This effect of cigarette is short lasting (36). On the other hand; smoking can cause depression and make it worse (36). 30% of smokers show some form of depression (37).

Sad patients may turn to nicotine as a way to regulate negative affect. Depressive symptoms positively correlated with current smoking and negatively correlated with likelihood of quitting smoking. Non

smokers are 51% less likely to be diagnosed with major depression than patients who had ever smoked. **G.Scott Acton et al** in their study found that `Depression is much more common in contemplation and preparation stages of quitting smoking than in pre-contemplation or maintenance stages (38)'.

Jim Rosack et al in their study found `Patients who have recovered from major depression and then stopped smoking have a greater likelihood of recurrence of depression on quitting tobacco (39)'.

They explained, Tyrosine hydroxylase when increased, lead to increased nor-epinephrine, which binds to α_2 receptors, whose function is to send signals from locus coeruleus to other parts of brain. In major depression, α_2 receptors and Tyrosine hydroxylase are increased. Chronic administration of anti-depressants decreases their levels. Smoking also decreases levels of α_2- receptors and tyrosine hydroxylase, thus having transient anti-depressant effect (39)'.

Janet Audrain et al in one study found that the HLA locus DRD 2A1 is associated with smoking progression and this is potentiated by depressive symptoms (40).

A common genetic defect could explain relation between depression and smoking as per **Harvard Medical School (37)**.

Nicotine acts at Acetyl choline receptor, thus stimulating release of dopamine, in brain's reward or motivation center. The brain becomes adapted to presence of the drug and no longer functions in absence of it. This will lead to depressive symptoms (37).

A study on 1000 people showed, depressed people were not more likely than controls to start smoking, but once started a history of daily smoking doubled the risk of developing major depression later on (37).

A survey of 3000 people in St. Louis area by **Josepha Cheong et al** for Psych Central found that life time prevalence of depression among smokers was 29% compared to 6.6% among non smokers.

Thus it has been found that smokers are more predisposed to depression. Depressed smokers are more likely to be unable to quit. So, use of anti-depressants during quitting doubles the rate of success of quitting.

COPD AND DEPRESSION

Burgass et al in the journal of Geriatrics state that, Depression occurs in 42% of patients with COPD. The lung disease may have a relatively poor long term outlook, leading to feelings of frustration, hopelessness and helplessness. Depressed mood lowers energy level further, making the symptoms even less tolerable (4)'.

Stage et al in Acta Psychiatrica Scandinavia found that, Depression in out-patients with COPD appears to be an independent predictor of mortality (41)'. Amongst COPD patients, severe COPD patients are at more risk of developing depression say **Van Maren et al in journal Thorax(42)**. In their study they found 25% depression in severe COPD compared to 19.6% in mild COPD and 17.6% in controls. Adjusting for other factors the risk of depression in COPD was 2.5 times that of controls.

Depression occurs in a higher frequency in any chronic condition but incidence of depression is found to be much higher in COPD than with other major chronic conditions. "Depression is more prevalent in people with COPD than in people with other chronic conditions, such as heart disease and even cancer" says **Dr. Rachel Norwood of the**

National Jewish Medical and Research Center in Denvel (43). Hypoxia due to COPD may contribute to depression (43). Also as breathing affects emotions, compromised breathing can be one reason for the increased prevalence of depression in COPD compared to other chronic conditions (43).

A study by **Florence et al (44)** showed that depression contributed to overall variance in functional status in COPD patients. Surprisingly COPD severity and disease burden did not contribute significantly to overall variance in functional status.

Among COPD patients, **Lin Mie et al** found a higher risk for depression among higher educated and higher income groups (45)

Kent Roundy et al in 102 COPD patients found that 50 had depression or anxiety but only 18 were found out in primary care (46).

Amongst 45 COPD patients assessed by **R.W.Light et al** in their study 42% had depression (47)

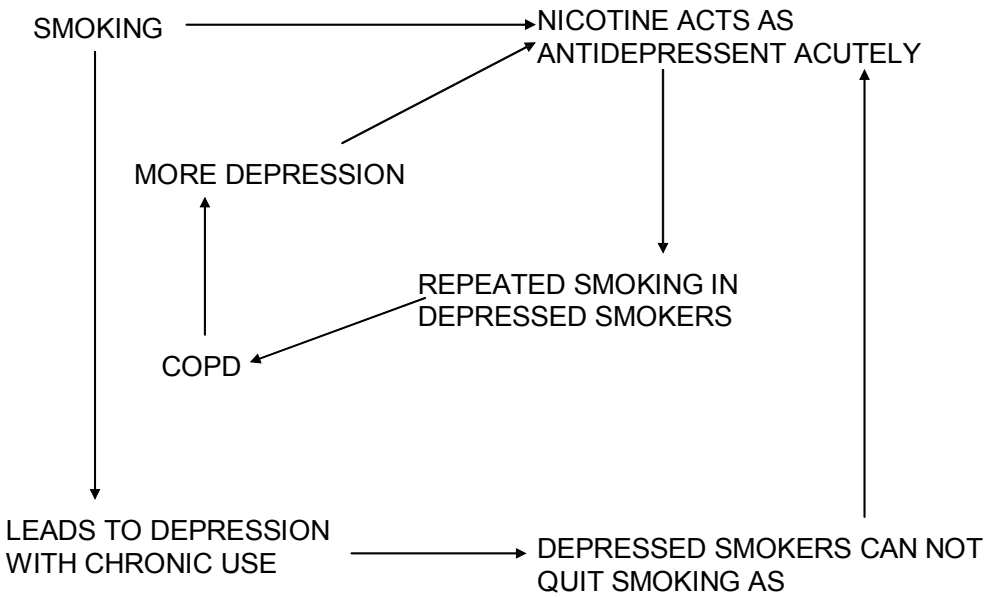
`Depression was the predominant emotional disturbance in COPD patients' observed **A.J.McSweeny et al** in their study (48).

Association between COPD and depression was noted in four controlled studies done by **L.Van Ede et al (49).**

Mark.E.Kunik et al found 80% of patients with chronic breathing disorders suffer from depression or anxiety but only 30% were receiving treatment (50).

Though there are many factors contributing to depression in COPD, the major one is smoking.

COPD, DEPRESSION AND SMOKING



A study by **Edwin.J.Wagena** showed that, Smoking people with chronic bronchitis or emphysema were more likely to report suffering from depressed mood when compared with smokers with no long lasting disease (Prevalence rate PR: 29.3 and 9.0% respectively . Odds Ratio (OR) for depressed mood = 4.04; 95% CI = 2.56 - 0.39) and when compared to smokers with a history of Heart disease, Hypertension or Myocardial Infarction (PR = 18.1%, OR = 1.99; 95%, CI = 1.07-3.68) or Rheumatoid arthritis (PR=20.1%, OR = 1.73; 95% CI = 0.96 - 3.11)' (51). COPD patients thus, have higher level of depression (52), which is increased by smoking (53).

Edwin.J.Wagena et al in Psychosomatic Medicine journal report a study where odds ratio for chronic bronchitis patients to have depression was 4.38 and the odds ratio for chronic bronchitis patients who smoke to have depression was 7.56(53).

Another study by **Edwin.J.Wagena** showed that, ` Several mechanisms may be responsible for the relation between smoking, depression and COPD. The physical illness itself keeps the patient depressed. Risk for depression increases with increasing severity of respiratory complaints. In smokers, the risk of depression is higher due to negative self evaluation (54)'.

Further more as smoking is related to depression by many ways (including genetic (55), neurotransmitter mediated (55), shared environmental factors as shown in a study by **Kendler** et al) (56), and COPD being a chronic disease can itself cause depression and as COPD is nearly always associated with smoking, it is not surprising to find that COPD is much more associated with depression that most other chronic diseases. So, it becomes mandatory for all of us to screen all COPD patients for depression and treat it as it will improve their quality of life.

Second and equally important is the fact that, As the single most important way of affecting outcome in all stages of COPD (57) and the only evidence based treatment (as confirmed by Lung Health study (58)) for preventing progression of COPD, quitting smoking is important. Depression is associated with failure to quit smoking and relapse. This is because although smoking causes depression over time, it has transient anti-depressant effect on brain and hence induces patients to relapse. This causes an increase in prevalence of depression by 25% in the first 6 months of quitting smoking. All these prevent a smoker with depression from quitting and thus a vicious cycle is established. Smoking causing depression and depression leading to smoking and

thus in long term smoking worsens COPD with its associated depressive symptoms.

Thus to break this cycle, an anti-depressant like bupropion should be given as the patient quits smoking. This will prevent relapse of smoking. This maintenance of quitting decreases COPD and hence depression also.

Furthermore distinguishing depression from COPD can be difficult as both are associated with same emotional & physical signs (59) (60). So, a formal screening for depression is of help to indicate whether anti-depressants are of use and in this study GDS-15 is used.

Thus COPD, smoking and depression are inter-related and breaking this chain of relation by using anti-depressants in needed patients, after a formal screen, can provide much improvement in quality of life of COPD patients.

Aim

AIM OF THE STUDY

- To compare the prevalence of depression between the elderly smoking COPD patients, healthy elderly smokers and healthy elderly controls.

- To assess the relationship between the severity of COPD and prevalence of depression.

Materials & Methodology

MATERIALS AND METHODS

This study was conducted in the Out Patient Clinic of the Department of Geriatric Medicine in the Government General Hospital attached to Madras Medical College, Chennai-03.

Collaborating Department

Department Of Thoracic Medicine in the Government General Hospital attached to Madras Medical College, Chennai-03.

Study Period

Seven months from February 2006 to August 2006.

Type of Study

Cross Sectional study

Ethical Committe Approval

Obtained.

Study Group

Total number of subjects studied was 90.They was divided into three groups of 30 subjects each.

Group A – COPD Patients who are also smokers

Group B – Healthy Smokers

Group C – Normal Healthy People

GROUP A

Inclusion criteria :

1) Age ≥ 60 yrs

2) Smoking bidis > 6 months , 15-30 bidis/day

3) Current smokers

4) Established users

5) Takes no other tobacco product (both smokeless and smoking varieties)

6) FEV_1 / FVC <70%. (Post-bronchodilator)

7) No other chronic disorder (Diabetes Mellitus/Ischemic Heart Disease/Rheumatoid Arthritis/ Seizures/Pulmonary Tuberculosis/Bronchial asthma / Osteo Arthritis / Low Back Ache/Hypertension/other chronic disorders)

8) Living with Family in harmony

9) Males

Exclusion Criteria

1) Age < 60 yrs

2) Non-smokers

3) Takes other smoking (or) smokeless tobacco other than beedis

4) Females

5) Any other chronic illness

6) Any acute illness (eg) Upper Respiratory Infections when the patient is having the illness before clinical cure

7) Living alone

8) People in old age homes

9) Post bronchodilator FEV_1/ FVC > 70%.

10) CXR showing finding suggestive of tuberculosis (or) history of pulmonary tuberculosis.

11) Asthma (> 12% reversibility of FEV1)

Thus Group A consists only of Smokers with COPD.

GROUP B

Inclusion criteria

1. Age ≥ 60 yrs
2. Smoking bidis > 6 months , 15-30 bidis/day
3. Current smokers
4. Established users
5. Takes no other tobacco product (both smokeless and smoking varieties)
6. FEV_1 / FVC >70%. (Post-bronchodilator)
7. No other chronic disorder.
8. Living with family in harmony.
9. Males

Exclusion Criteria:

1. Age < 60 yrs
2. Non-smokers
3. Takes any other smoking (or) smokeless tobacco other than beedis.
4. Females
5. Any other chronic illness

6. Any acute illness (eg) URI when the patient is having the illness before clinical cure
7. Living alone
8. People in old age homes
9. Post bronchodilator FEV_1/ FVC < 70%.
10. CXR showing finding suggestive of tuberculosis (or) H/O pulmonary tuberculosis
11. Asthma associated. Reversibility of FEV1>12%.

Thus Group B consists only of Smokers who are healthy otherwise.

GROUP C

Inclusion Criteria :

1. Age ≥ 60 yrs
2. Non-smokers / Not taking any form of tobacco
3. FEV_1/FVC > 70%. Post bronchodilator.
4. Living with family in harmony.
5. Males

Exclusion Criteria :

1. Age < 60 yrs
2. Female
3. Any chronic illness
4. Any acute illness before clinical cure
5. Living alone
6. Tobacco use in any form.

In all three groups, all subjects who were included were derived from low income house holds (<40,000 rupees per year). Every one of them belonged to Occupation category-5 (unskilled laborers) .This was done to bring about equality among all three groups in socio-economic status and also because they form the majority of patients attending the OPD.

Healthy Smokers and Non Smokers were selected from those coming for routine check up in Geriatric Medicine OPD.

Study Method

Informed consent was obtained from all subjects entering study. History was obtained to rule out any other chronic and acute diseases given in exclusion criteria. Chest X ray was taken to

rule out tuberculosis and other pulmonary disorders. ECG was done to rule out primary cardiac disorders. Blood urea, sugar, creatinine and electrolytes had to be normal for subjects to enter the study.

History of smoking was obtained and subjects were divided into two sets based on it.

Set 1 - Smokers

Set 2 - Non Smokers

Among set-1 only those who satisfy following criteria were selected

1. Beedis only
2. No other smoking (example –cigarette, cigar, etc)/smokeless tobacco being used
3. current smokers
4. 16-30 beedis per day and so automatically, `established smokers'.

Others were excluded from set -1.

In set-2 only those who had never had even a puff of smoking history and having no history of using smokeless tobacco were included.

Spirometry was done in both sets. If there was a reversibility of FEV1 of more than 12%, the subjects were excluded. The remaining subjects were stratified, as per FEV1 /FVC.

- If FEV1 /FVC <70% in set-1= labeled as smokers with COPD.(GROUP-1)
- If FEV1 /FVC >70% in set-1= labeled as Healthy smokers. (GROUP-2)
- If FEV1 /FVC >70% in set-2= labeled as Healthy controls. (GROUP-3)
- If FEV1 /FVC < 70% in set-2= excluded from the study.

Now group - 1 was subdivided into four subgroups as per GOLD criteria, using FEV1 %.

Subgroups	Post bronchodilator FEV1 %
1	≥ 80%
2	≥ 50% - < 80%
3	≥ 30% - < 50%
4	< 30%

All subjects from three groups were administered Geriatric depression scale – 15 version which has been validated in the Indian elderly.

A score of more than or equal to 5 was taken as positive for depression and a score of less than 5 was taken as negative for depression.

Results were obtained and analyzed statistically.

Methods used in analysis

1. One way ANOVA F test followed by Bonferroni t –test was used to compare.

    a. mean ages of all three groups ,

b. FEV1/FVC of all three groups,

c. mean heights of all three groups

d. GDS scores amongst three groups

e. GDS scores amongst four subgroups of group 1.

2. Fischer Exact Test was used to compare GDS Results of all three groups

3. Chi Square test was used to compare results of GDS amongst four subgroups of group 1.

Results

RESULTS

TABLE 1

AGE

GROUP	AGE		
	MEAN	**SD**	**p value**
PATIENTS	69.90	6.40	0.6671 (N.S)
HEALTHY SMOKERS	69.80	7.53	
CONTROLS	68.43	7.13	

Using One Way ANOVA Age has p = 0.6671 (not significant)

Multiple Range Tests : Modified LSD (Bonferroni) test with significance level .05

Harmonic Mean Cell size = 30.0000

The actual range used is the listed RANGE * 1.2849 with the following value(s) for RANGE: 3.45

No two groups are significantly different at the .050 level

Homogeneous Subsets (highest and lowest means are not significantly different)

Subset 1

Group	Group 3	Group 2	Group 1
Mean	68.4333	69.8000	69.9000

TABLE 2

HEIGHT

GROUP	HEIGHT		
	MEAN	**SD**	**p value**
PATIENTS	161.63	4.81	0.626 (N.S)
HEALTHY SMOKERS	162.1	4.23	
CONTROLS	162.8	4.99	

Using One Way ANOVA Height has p = 0.626 (not significant)

Multiple Range Tests : Modified LSD (Bonferroni) test with significance level .05

Harmonic Mean Cell size = 30.0000

The actual range used is the listed RANGE * .8556 with the following value(s) for RANGE: 3.45

No two groups are significantly different at the .050 level

Homogeneous Subsets (highest and lowest means are not significantly different)

Subset 1

Group	Group 1	Group 2	Group 3
Mean	161.6333	162.1000	162.8000

TABLE 3

RATIO OF FEV1/FVC

GROUP	FEV1/FVC		p value
	MEAN	SD	
PATIENTS	54.3333	7.07	< 0.001
HEALTHY SMOKERS	75.8000	2.47	
CONTROLS	78.0667	3.20	

Using One Way ANOVA FEV1/FVC has $p < 0.001$ (significant)

Multiple Range Tests : Modified LSD (Bonferroni) test with Significance level .05

Harmonic Mean Cell size = 30.0000

The actual range used is the listed RANGE * .8584 with the following value(s) for RANGE: 3.45

(*) Indicates significant differences which are shown in the lower triangle

Mean	GROUP	
54.3333	Group 1	
75.8000	Group 2	*
78.0667	Group 3	*

Thus Groups 2 and 3 do not differ significantly.

Homogeneous Subsets (highest and lowest means are not significantly different)

Subset 1

Group	Group 1
Mean	54.3333

Subset 2

Group	Group 2	Group 3
Mean	75.8000	78.0667

TABLE 4

GDS SCORES IN GROUPS

GROUP	GDS SCORES		p value
	MEAN	SD	
PATIENTS	8.27	4.57	< 0.001
HEALTHY SMOKERS	5.17	3.96	
CONTROLS	2.57	2.70	

Using One Way ANOVA GDS Scores have $p < 0.001$ (significant)

Multiple Range Tests : Modified LSD (Bonferroni) test with significance level .05

Harmonic Mean Cell size = 30.0000

The actual range used is the listed RANGE * .6979 with the following value(s) for RANGE: 3.45

(*) Indicates significant differences which are shown in the lower triangle

Mean	GROUP	
2.5667	Group 3	
5.1667	Group 2	*
8.2667	Group 1	* *

Homogeneous Subsets (highest and lowest means are not significantly different)

Subset 1

Group	Group 3
Mean	2.5667

Subset 2

Group	Group 2
Mean	5.1667

Subset 3

Group	Group 1
Mean	8.2667

TABLE 5

GDS SCORES IN SUB GROUPS

SUB GROUP	GDS SCORES		p value
	MEAN	SD	
STAGE 1	2.50	0.71	0.123
STAGE 2	7.92	4.66	
STAGE 3	8.58	4.52	
STAGE 4	12.33	0.58	

Using One Way ANOVA GDS SCORES have p =0.123 (not significant)

Multiple Range Tests : Modified LSD (Bonferroni) test with significance level .05

Harmonic Mean Cell size = 4.0258

The actual range used is the listed RANGE * 2.1570 with the following value(s) for RANGE: 4.04

(*) Indicates significant differences which are shown in the lower triangle.

Mean STAGE

2.5000 Stage1

7.9231 Stage 2

8.5833 Stage 3

12.3333 Stage 4 *

Homogeneous Subsets (highest and lowest means are not significantly different)

Subset 1

Group	Sub Group 1	Sub Group 2	Sub Group 3
Mean	2.5000	7.9231	8.5833

Subset 2

Group	Sub Group 2	Sub Group 3	Sub Group 4
Mean	7.9231	8.5833	12.3333

This shows Sub Groups 1 and 4 differ significantly from each other but not others.

TABLE 6

GDS RESULT IN GROUPS

GDS RESULT	POSITIVE	NEGATIVE	PERCENTAGE OF POSITIVES
PATIENTS	19	11	63.3
SMOKERS	9	21	30
CONTROLS	2	28	6.7

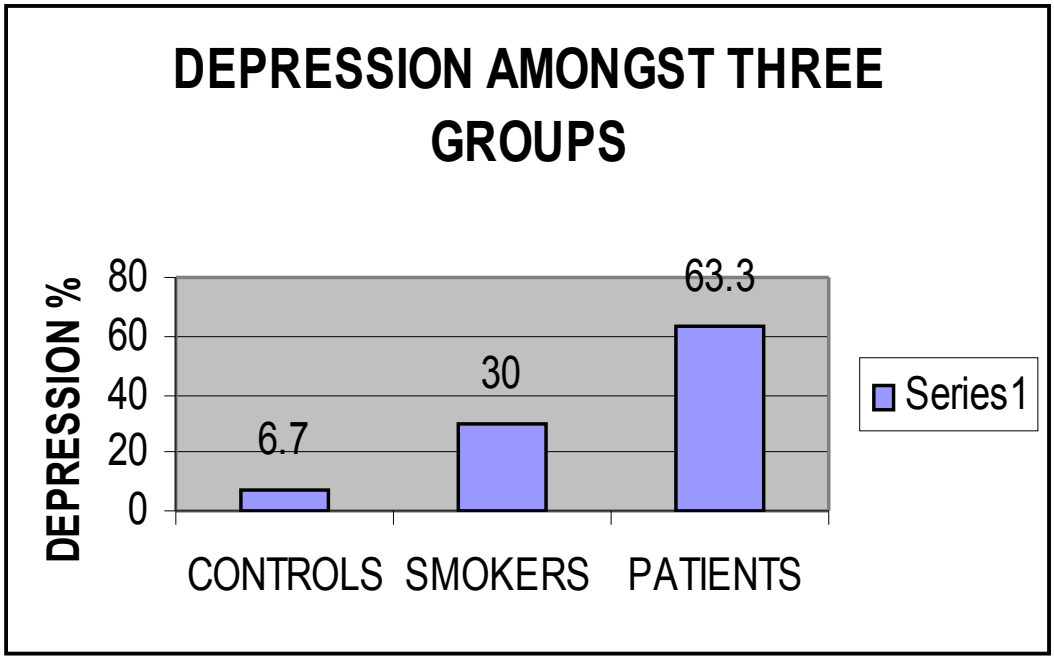


TABLE 7a GDS RESULT IN GROUPS

COPD PATIENTS AND HEALTHY CONTROLS -

GROUP	DEPRESSED	NOT DEPRESSED	p VALUE
COPD	19	11	p <0.001
CONTROLS	2	28	

Fischer's Exact Test used. p <0.001(very significant)

TABLE 7b

COPD PATIENTS AND HEALTHY SMOKERS

GROUP	DEPRESSED	NOT DEPRESSED	p VALUE
COPD	19	11	0.0191
HEALTHY SMOKERS	9	21	

Fischer's Exact Test used. p = 0.0191 (significant)

TABLE 7c

HEALTHY SMOKERS AND CONTROLS

GROUP	DEPRESSED	NOT DEPRESSED	p VALUE
HEALTHY SMOKERS	9	21	0.0419
CONTROLS	2	28	

Fischer's Exact Test used. p = 0.0419 (significant)

TABLE 8

GDS RESULTS IN SUB GROUPS

GDS RESULT	STAGE 1	STAGE 2	STAGE 3	STAGE 4	p value
POSITIVE	0	8	8	3	0.15327
NEGATIVE	2	5	4	0	

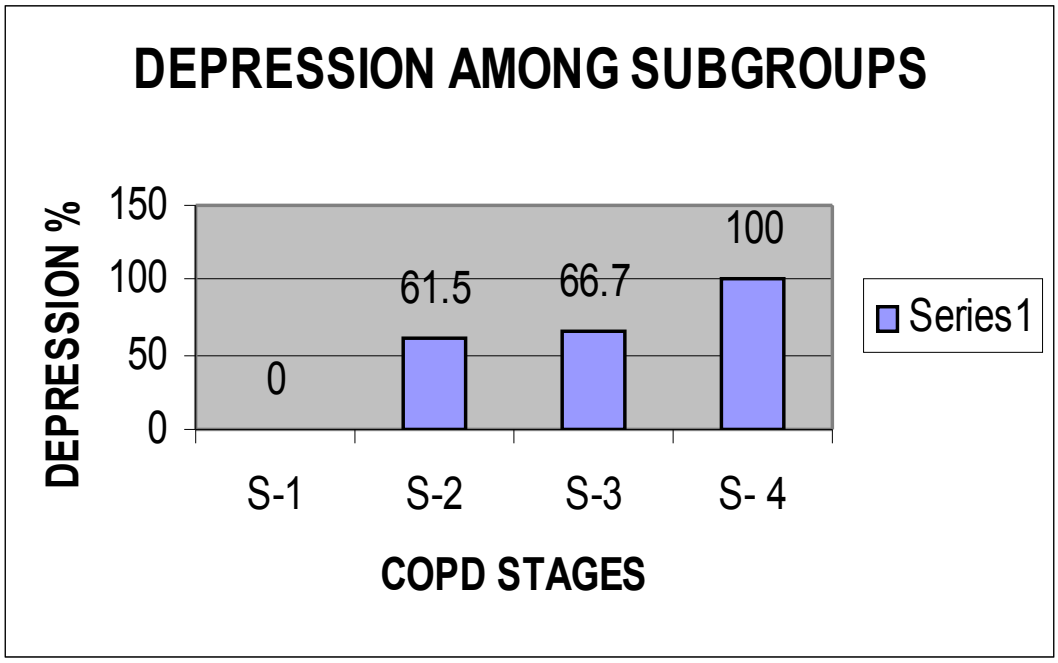
Chi-Square	Value	DF	Significance
Pearson	5.2664	3	0.15327

Using Chi-Square test p = 0.15327 (not significant).

TABLE 9

GDS RESULTS IN SUB GROUPS

GDS RESULT	POSITIVE	NEGATIVE	PERCENTAGE OF POSITIVES
STAGE 1	0	2	0
STAGE 2	8	5	61.5
STAGE 3	8	4	66.7
STAGE 4	3	0	100



Discussion

DISCUSSION

This study was done to find out the association between COPD, depression and smoking in elderly people. Here 90 subjects were divided into three groups of 30 each.

Group 1 - COPD patients who are also smokers.

Group 2 - Healthy smokers.

Group 3 - Healthy controls

Group 1 was subdivided into four sub groups.

Sub group 1 - FEV1 $\geq$ 80%

Sub group 2 - FEV1 $\geq$ 50% to <80%

Sub group 3 - FEV1 $\geq$ 30% to <50%

Sub group 4 - FEV1 < 30%

In all these volunteers, Spirometry and Geriatric Depression Scale were administered, along with an enquiry about their smoking patterns. Statistical analysis was done and results were compared.

AGE

Age affects depression and so, comparison between groups of different Ages will bias the study.

To eliminate bias, age was compared in all three groups using one way ANOVA followed by Modified LSD (Bonferroni) test. p value of 0.6671 was obtained. This is not significant. So, `Age' as a confounding factor was ruled out.

HEIGHT

Using one way ANOVA, followed by Modified LSD (Bonferroni) test, Height was compared in all three groups. p value of 0.626 was obtained. This is not significant. So, `Height' as a confounding factor was ruled out.

FEV1/FVC

FEV1/FVC was used to separate Group 1 from other two groups. FEV1/FVC < 70% indicates COPD and this is internationally accepted definition (15). So, it was just enough to prove that FEV1/FVC does not vary between Groups 2 and 3. By use of Modified LSD (Bonferroni) test it was found that the means of Groups 2 (75.8000) and Group 3 (78.0667) do not differ significantly. Thus the role of FEV1/FVC as a confounding factor in non-COPD groups was ruled out.

GDS SCORES

GDS scores were given to an individual person based on fifteen questions. A person can have any score between zero to fifteen.

GDS scores were compared between all three groups using one way ANOVA, followed by Modified LSD (Bonferroni) test. Significant difference was found between all three groups ($p < 0.001$) and each group differed from each other significantly.

GDS scores were compared between all four sub-groups of Group-1 using one way ANOVA, followed by Modified LSD (Bonferroni) test. Significant difference was not found between all four sub-groups ($p = 0.123$). However two homogenous sub sects occurred (sub-groups 1,2,3 and sub-groups 2,3,4).This shows that sub-group1 varied from sub-group 4 but other sub- groups did not differ from each other significantly.

GDS RESULTS

Though GDS scores were compared, comparison of results (positive or negative) is much more important.

Geriatric Depression Scale was specifically developed to find out depression in elderly, as depression in elderly manifests differently from that of younger age group. Elderly have more somatic complaints due to depression compared to young patients. The original version of Geriatric Depression Scale consists of 30 parameters .Here Normal score is 5 (plus or minus 4). Patients with mild depression have a score of 15 (plus or minus 6) and patients with severe depression have a score of 23 (plus or minus 5). . **Ada C Mui et al** in their study have found that GDS is valid in Indian Population (35).

A Shorter Version, Geriatric Depression Scale – 15, is often used for ease of administration. Sheikh & Yesavage had recommended use of GDS in Gerontology journal as early as in 1986 (33). This short version is considered positive if score is 5 or more out of 15. This short version is recommended in Current Geriatric Diagnosis and Treatment 2004 edition. Thus as per this there can be only two results – Positive or Negative.

GDS SCORE	DEPRESSION
0 - 4	Positive
5 - 15	Negative

This shorter version of GDS was used in this study.

In this study prevalence of depression

- Among smoking COPD patients was 63.3%.
- Among healthy smokers was 30% and
- Among healthy controls was 6.7%.

In the current study, it has been found that the prevalence of depression

- Between elderly COPD patients who smoke and healthy elderly non smokers was highly statistically significant ($p < 0.001$).
- Between the elderly COPD patients who smoke and healthy elderly smokers ($p = 0.0191$) was statistically significant.
- Between healthy elderly smokers and healthy elderly non smokers ($p = 0.0419$) was also statistically significant.

This is a very valid observation considering the fact that most COPD patients in the community are not screened for depression and also due to the fact that depression more than adds to the morbidity caused by COPD.

This study also observes that the disease `COPD' per se can cause depression as indicated by an increased prevalence of depression among smoking COPD patients compared to healthy smokers.

This study also points out that smoking is associated with one more problem that is rarely addressed to – that is, Smokers are more likely to be depressed. This assumes significance considering the fact that depressed smokers are less likely to quit smoking, compared to non depressed ones. Since quitting smoking reduces lots and lots of morbidity and mortality, it becomes a necessity for the Medical Community to come up with ways that will increase the success of quitting. Since `depression' prevents quitting, a formal screen for depression and treatment with antidepressants will enhance the success of quitting. This view is supported by the recommendation of the anti-depressant bupropion for quitting by FDA and its success in this situation.

The results thus show that COPD per se can cause depression and when smoking adds to it, the prevalence is much higher than in healthy non smokers. Since most COPD patients are smokers, the above observation goes on to stress that – Screening for Depression is a sin qua non in all COPD patients.

In this study prevalence of depression among subgroups of smoking COPD patients were 0% in stage 1 COPD, 61.5% in stage 2 COPD, 66.7% in stage 3 COPD and 100% in stage 4 COPD. GDS results were compared applying Chi-Square test and was found to be insignificant (p =0.15377) amongst all four sub-groups. Even though the comparison between the stages of COPD and GDS results were not statistically significant, it has been found that the severe COPD patients had more prevalence of depression compared to milder groups.

HEALTHY SMOKERS AND CONTROLS

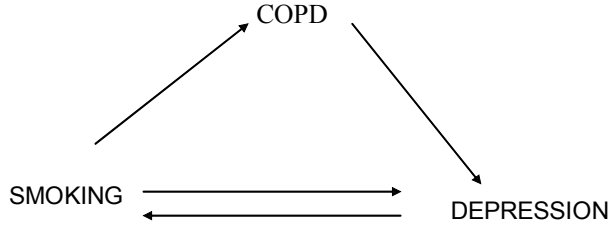
Thus this study observed a significant increase in the prevalence of depression amongst healthy smokers compared to healthy controls. This finding correlates with a survey reported in Psychcentral of 3,000 individuals in the St. Louis area which confirmed that lifetime frequency of major depression was more common among smokers than nonsmokers (6.6 vs. 2.9 percent).

This also correlates with the findings of a study reported by **Harvard Medical School** on 1000 people which showed depressed people were not more likely than controls to start smoking but once

started, a history of daily smoking doubled the risk of developing major depression later on (37).

This also correlates with the findings of a study reported by **Stephen J. Jay MD among** persons 65 years of age or older; the smoking rate in that study was reported as 12.9% among women and 21.2% among men. Smoking was a major risk factor for 7 of the 14 leading causes of death among persons 60 years of age and older and was a complicating factor for 3 others in that study. Thirty percent to 50% of smokers had a history of depression. Thus, that study concluded that, `smoking is not uncommon in the elderly and is associated with both depression and numerous adverse health effects, including illness, death, reduced health care utilization, reduced physical activity, and altered metabolism of medications (61)'The Present study correlates closely with this above mentioned study with a depression rate among smokers coming to 30%.

HEALTHY SMOKERS AND COPD PATIENTS WHO SMOKE



As already mentioned, this study also observed a significant increase in the prevalence of depression among COPD patients who smoke compared to healthy smokers.

This correlates with the study by **Edwin.J.Wagena** (51) which showed that, Smoking people with chronic bronchitis or emphysema were more likely to report suffering from depressed mood when compared with smokers with no long lasting disease (Prevalence rate PR: 29.3 and 9.0% respectively. Odds Ratio, OR for depressed mood = 4.04; 95% CI = 2.56 - 0.39).

This also correlates with the findings of another study reported by **Edwin.J.Wagena et al in Psychosomatic Medicine journal** where odds ratio for chronic bronchitis patients to have depression was 4.38 and the odds ratio for chronic bronchitis patients who smoke to have depression was 7.56(53).

COPD PATIENTS WHO SMOKE AND HEALTHY CONTROLS

As already mentioned, this study also observed a very significant difference in the prevalence of depression among COPD patients who smoke compared to healthy non-smoking controls.

This is in correlation with studies of **L.Van Ede et al (49)**who found an increase in depression among COPD patients compared to controls in three of his studies. Two other studies by of L.Van Ede et al (49) which did not show a significant difference in prevalence of depression between COPD patients and controls were excluded by the Author himself as he found that those studies had defective methodology. He then showed an increased prevalence of depression COPD patients in general population in three of six non controlled studies.

This is also in correlation with the study of ABEBW **M.YOHANNES** (62)who compared the prevalence of depressive symptomatology in elderly outpatients with stable COPD with that in healthy controls and age-matched patients with other disabilities, and also assessed the relation between degree of disability, quality of life and depressive symptoms. The subjects were 96 older people with COPD [56 men; aged 70–93 (mean 78) years], 55 normal controls [23 men; aged 70–90 (mean 78) years] and 53 disabled controls [27 men; aged 70–92 (mean 78) years]. Exclusion criteria were acute respiratory exacerbation or use of oral steroids in the last 6 weeks, known previous psychiatric disorder and acute or chronic confusion. The study found

that depressive symptoms are common in elderly patients with COPD; prevalence and/or severity of depressive symptoms may be greater in those who are most disabled. In this study also similar results were obtained with increase in prevalence of depression among COPD patients with a much higher prevalence in Stage 4 disease.

This study also is in correlation with a study reported by **A J McSweeny et al in Archieves of Internal Medicine (48)**.In that study 203 patients with COPD and 73 controls were selected. Depression was the predominant emotional disturbance noted in COPD patients.

According to a recent survey by Mark Kunic and others, anxiety and depressive disorders were found in 60 to 80 percent of military veterans with COPD. Incidence of smoking and mood disorders is significantly higher in VA clinic patients (50).Thus the present study correlates with the study of **Mark Kunic et al** in that in this study also the prevalence of depression is 63%

This incidence of depression among COPD patients in this study is however higher than the study by **Kent Roundy et** al (In 102 COPD patients found that 50 had depression or anxiety) (46). This is also higher than the study by **R.W.Light et al** who used Beck Depression

Inventory in 45 patients and found 42% of them had depression (47). This may be due to the fact that all patients who entered this study were of lower socio-economic status and this could have increased the prevalence of depression equally in all three groups.

DEPRESSION AMONG DIFFERENT STAGES OF COPD -

This study observed an increase in the prevalence of depression among COPD patients of Stage 4 compared to Stage 1. The prevalence of depression increases from 0% in Stage 1 to 61.5% in Stage 2 to 66.7% in Stage 3 and becomes 100% in Stage 4. Though there is an increase in the prevalence of depression with increasing stage of COPD, statistically significant increase between all stages is not found. This contrasts with a study by **Van Manen et al in journal Thorax (42)**. In their study they found 25% depression in severe COPD compared to 19.6% in mild COPD and 17.6% in controls. This difference between the two studies might have been due to the smaller sample size used in this study. However this study correlates with the study of Van Manen et al in that prevalence of depression increases with increasing severity in both studies.

LIMITATIONS OF THIS STUDY

In this study COPD patients without a history of smoking were not included due to the difficulty in getting such patients. Only a few of them came to Out Patient Department (as they constitute only a minority of COPD patients) and nearly every one of them had other predispositions for depression and so could not enter the study. Had this study included them, this study would have been of more value. Still, as most COPD patients in the community are smokers, this study represents the bulk of COPD patients in the community and their predisposition for depression.

The stages of COPD were not equally represented in this study. Most patients were of Stage 2 or 3. This may be due to the fact that most if not all patients of Stage 1 have no symptoms and so rarely come to OPD for treatment. Most patients in Stage 4 may be disabled secondary to their lung disease and many of them may be bed ridden, either admitted in ward or being treated at home itself and so they also form only a small percentage of out patients. These problems of this study could have been solved, had the study been carried out in the community rather than in the hospital.

Future studies are needed to find out how many COPD patients with depression are really being treated for their depression. Also studies are needed to prove that antidepressants really help COPD patients and improve their morbidity and mortality.

Summary

SUMMARY

This study was done to find out the correlation between COPD, Smoking and Depression. Three Groups of subjects were selected with 30 subjects in each Group. Group 1 consisted of COPD patients with every one of them being smokers. Group 2 consisted of healthy smokers. Group 3 consisted of healthy controls. Group 1 was sub divided into four sub groups based on the severity of COPD (FEV1%).All FEV1 values were post bronchodilator values (63).

All subjects were administered Spirometry and Geriatric Depression Scale – 15 version along with a detailed history of smoking.

Results obtained were subjected to statistical analysis. It was found that COPD patients who smoke were more depressed than healthy smokers, who in turn were more depressed than healthy controls. This study also observed an increase in the percentage of depressed patients with increasing severity of COPD, though that increase did not reach statistical significance.

Thus this study observes a very high prevalence of depression in COPD patients who smoke compared to healthy controls. This study concludes that, **all COPD patients should be screened for depression.**

Conclusion

CONCLUSION

The prevalence of depression was evaluated in COPD patients and healthy smokers. This study shows

- Elderly COPD patients who also smoke have a very high prevalence of depression compared to healthy elderly non smokers.

- Elderly smokers with COPD have higher prevalence of depression compared to elderly healthy smokers.

- Elderly healthy smokers have higher prevalence of depression than healthy elderly non smokers.

- Prevalence of depression increases with increasing stage of COPD but not to a significant extent.

Annexure I

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Master

Charts

SL. No.	GROUP I	O.P. No.	AGE	HEIGHT	FEV1 PREDICTED	FVC PREDICTED	FEV1 OBSERVED	FVC OBSERVED	FEV1/ FVC	GDS SCORE	RESULT
P1	Vadivel	417/06	65	155	1.99	2.52	1.59	2.33	68	2	N
P2	Manickam	1132/05	63	160	2.23	2.82	1.69	2.64	64	2	N
P3	Devandran	608/06	67	167	2.32	2.98	1.86	2.81	66	3	N
P4	Kathirvel	654/04	70	160	2.05	2.66	1.39	2.44	57	3	N
P5	Babu	280/03	72	165	2.19	2.86	1.23	1.98	62	12	P
P6	RajaPillai	42/04	75	165	2.11	2.80	1.31	2.30	57	11	P
P7	Gopalan	1034/03	80	165	1.99	2.68	1.09	2.22	49	12	P
P8	Venkatesan	689/04	60	160	2.31	2.88	1.20	2.07	58	3	N
P9	Annamalai	766/06	66	172	2.60	3.35	1.95	3.25	60	11	P
P10	Chellan	123/04	67	163	2.24	2.88	1.19	2.48	48	12	P
P11	Thomas	423/05	68	167	2.37	3.05	1.21	2.50	49	3	N
P12	Seeman	1320/05	67	160	2.13	2.73	1.41	2.71	52	9	P
P13	Boobalan	78/04	69	164	2.23	2.88	1.43	2.65	54	11	P
P14	Shanmugam	321/02	71	157	1.92	2.49	1.29	2.35	55	1	N
P15	Kali	987/04	74	170	2.33	3.07	1.28	2.78	46	13	P
P16	Danasekaran	543/03	61	156	2.13	2.66	1.00	1.85	54	12	P
P17	Siva	241/05	77	153	1.69	2.25	0.64	1.56	41	11	P
P18	Sunderasan	432/04	74	160	1.95	2.57	0.72	1.80	40	2	N
P19	Antony	564/02	79	163	1.94	2.61	0.68	1.26	54	10	P
P20	Prakasam	865/05	77	158	1.80	2.40	0.77	1.33	58	4	N
P21	Baskaran	786/03	77	164	2.03	2.70	0.81	1.35	60	12	P
P22	Iyyappan	510/04	78	159	1.81	2.43	0.56	0.97	58	II	P
P23	AllaPitchai	126/01	78	167	2.11	2.83	0.57	1.10	52	12	P
P24	ThambiRaj	43/03	61	164	2.43	3.06	0.70	1.48	47	13	P
P25	Palaniappan	622/05	61	154	2.09	2.61	0.59	1.11	53	12	P
P26	Munusamy	765/02	68	161	2.14	2.75	0.73	1.18	62	3	N
P27	Seenivasan	564/04	72	162	2.08	2.71	0.79	1.80	44	15	P
P28	MahaRajan	542/01	63	165	2.42	3.07	1.04	2.17	48	10	P
P29	Kannappan	894/05	77	153	1.69	2.25	0.83	1.54	54	2	N
P30	Sahul	542/06	60	160	2.31	2.88	0.95	1.58	60	11	P

SL. No.	GROUP II	O.P. No.	AGE	HEIGHT	FEV1 PREDICTED	FVC PREDICTED	FEV1 OBSERVED	FVC OBSERVED	FEV1/ FVC	GDS SCORE	RESULT
S1	Radhakrishnan	330/06	60	170	2.68	3.39	2.14	2.93	73	2	N
S2	Gangadaran	234/06	63	165	2.42	3.07	2.08	2.77	75	0	N
S3	Munusamy	143/06	79	157	1.71	2.30	1.45	1.89	77	3	N
S4	Subramoni	220/06	65	160	2.18	2.77	1.81	2.42	75	4	N
S5	Kuppusamy	562/06	74	164	2.10	2.77	1.81	2.18	83	10	P
S6	Alagesan	321/06	60	156	2.16	2.68	1.81	2.39	76	4	N
S7	Govindan	663/06	71	163	2.14	2.79	1.84	2.45	75	4	N
S8	Mani	231/06	78	162	1.93	2.58	1.74	2.26	77	11	P
S9	Ramasamy	169/06	77	158	1.80	2.40	1.49	1.89	79	1	N
S10	Perumal	612/06	68	161	2.14	2.75	1.73	2.34	74	4	N
S11	Damodaran	320/06	63	160	2.23	2.82	2.05	2.74	75	11	P
S12	Abdul	280/06	60	160	2.31	2.88	1.94	2.66	73	2	N
S13	Arumugam	345/06	79	169	2.16	2.91	1.88	2.51	75	9	P
S14	Kannan	730/06	61	154	2.09	2.61	1.76	2.37	74	2	N
S15	Murugesan	432/06	78	159	1.81	2.43	1.54	2.08	74	11	P
S16	Chellappan	881/06	78	167	2.11	2.83	1.79	2.46	73	11	P
S17	Ponnusamy	465/06	60	156	2.16	2.68	1.86	2.41	77	4	N
S18	Samy	423/06	78	167	2.11	2.83	1.77	2.36	75	14	P
S19	Ragavan	876/06	79	160	1.83	2.46	1.46	1.98	74	3	N
S20	Sami	795/06	67	160	2.13	2.73	1.83	2.51	73	2	N
S21	Madavaraj	154/06	72	162	2.08	2.71	1.75	2.24	78	3	N
S22	Mohd.Bazir	397/06	79	160	1.83	2.46	1.54	1.92	80	2	N
S23	Jayaraman	112/06	79	169	2.16	2.91	1.90	2.38	80	3	N
S24	Ganasan	203/06	63	163	2.34	2.97	1.99	2.69	74	12	P
S25	Duraisamy	814/06	67	160	2.13	2.73	1.79	2.35	76	2	N
S26	Saminathan	734/06	60	170	2.68	3.39	2.41	3.30	73	8	P
S27	Farook	510/06	67	160	2.13	2.73	1.83	2.32	79	4	N
S28	Sambasivam	208/06	77	164	2.03	2.70	1.68	2.22	76	3	N
S29	Palani	176/06	61	164	2.43	3.06	2.11	2.82	75	2	N
S30	Krishnan	312/06	71	163	2.14	2.79	1.75	2.31	76	4	N

SL. No.	GROUP III	O.P. No.	AGE	HEIGHT	FEV1 PREDICTED	FVC PREDICTED	FEV1 OBSERVED	FVC OBSERVED	FEV1/ FVC	GDS SCORE	RESULT
C1	Dilli	215/06	60	165	2.52	3.16	2.42	3.14	77	1	N
C2	Gopalan	532/06	78	165	2.04	2.73	1.94	2.55	76	10	P
C3	Muniandi	419/06	63	170	2.60	3.32	2.34	3.12	75	0	N
C4	Manikannan	365/06	65	165	2.32	3.02	2.30	2.77	83	2	N
C5	Ammavasai	254/06	73	159	1.94	2.54	1.82	2.28	80	3	N
C6	Elumalai	450/06	67	158	2.05	2.63	1.97	2.43	81	2	N
C7	Arunachalam	621/06	71	170	2.40	3.14	2.14	2.67	80	4	N
C8	Govindasamy	524/06	77	170	2.25	3.00	2.27	2.91	78	2	N
C9	Chandrasekaran	709/06	60	156	2.16	2.68	1.99	2.42	82	3	N
C10	Raj	604/06	78	167	2.11	2.83	1.92	2.46	78	2	N
C11	Mohan	274/06	71	163	2.14	2.79	2.03	2.68	76	4	N
C12	Nadaraj	711/06	79	160	1.83	2.46	1.65	2.06	80	13	P
C13	Muthukrishnan	479/06	65	160	2.18	2.77	1.98	2.54	78	1	N
C14	Christopher	583/06	61	156	2.13	2.66	2.00	2.36	85	2	N
C15	Hari	716/06	79	169	2.16	2.91	2.18	2.95	74	3	N
C16	Rangiah	178/06	60	165	2.52	3.16	2.29	3.14	73	2	N
C17	Vaelu	771/06	67	158	2.05	2.63	1.93	2.57	75	1	N
C18	Arjunan	498/06	77	170	2.25	3.00	2.12	2.75	77	3	N
C19	Srinivasan	698/06	65	165	2.32	3.02	2.13	2.70	79	2	N
C20	Varadan	297/06	67	160	2.13	2.73	2.04	2.49	82	1	N
C21	Chari	680/06	60	170	2.68	3.39	2.55	3.44	74	2	N
C22	Keasavan	371/06	60	160	2.31	2.88	2.24	2.87	78	0	N
C23	Mohd.Ali	727/06	67	160	2.13	2.73	1.98	2.61	76	4	N
C24	Sankar	596/06	71	163	2.14	2.79	1.93	2.38	81	3	N
C25	Baasha	654/06	61	156	2.13	2.66	1.96	2.33	84	1	N
C26	Vasudevan	387/06	77	153	1.69	2.25	1.52	1.98	77	1	N
C27	Chitiah	744/06	79	157	1.71	2.30	1.62	2.23	73	2	N
C28	SultanMohd.	190/06	74	164	2.10	2.77	1.83	2.44	75	1	N
C29	Ethiraj	820/06	61	164	2.43	3.06	2.21	2.84	78	2	N

C30	Dharman	823/06	60	166	2.61	3.25	2.48	3.22	77	0	N

Annexure

II

PROFORMA

Name – Age -

Sex – Height -

Address – O.P.Number-

Short History of Present Illness - Care Giver Support -

Smoking History –

Co-morbid Illness –

Geriatric Depression Scale score –

FEV1 Predicted - FEV1 Reversibility–

Post Bronchodilator FVC Observed – FVC Predicted -

Post Bronchodilator FEV1 Observed –

FEV1/FVC – FEV1% -

GROUP – Smoking COPD patients/ Healthy smokers/Controls

Sub Group (if COPD patient) –

Geriatric Depression Scale Result – Positive / Negative

GERIATRIC DEPRESSION SCALE(GDS) SHORT FORM

HOW YOU FELT THIS PAST WEEK

Are you basically satisfied with your life?	yes	**NO**
Have you dropped many of your activities and interests?	**YES**	no
Do you feel that your life is empty?	**YES**	no
Do you often get bored?	**YES**	no
Are you in good spirits most of the time?	yes	**NO**
Are you afraid that something bad is going to happen to you?	**YES**	no
Do you feel happy most of the time?	yes	**NO**
Do you often feel helpless?	**YES**	no
Do you prefer to stay at home, rather than going out and doing new things?	**YES**	no
Do you feel you have more problems with memory than most?	**YES**	no
Do you think it is wonderful to be alive now?	yes	**NO**
Do you feel pretty worthless the way you are now?	**YES**	no
Do you feel full of energy?	yes	**NO**
Do you feel that your situation is hopeless?	**YES**	no
Do you think that most people are better off than you are?	**YES**	no

Score: _____ (Number of "depressed" answers - ones that are **bold**)

Answers in **bold** indicate depression.

ABBREVIATIONS

1. COPD – Chronic Obstructive Pulmonary Disease
2. GOLD – Global Initiative Of Obstructive Lung Diseases
3. ATS – American Thoracic Society
4. GDS – Geriatric Depression Scale
5. ERS – European Respiratory Society
6. FEV1 – Forced Expiratory Volume In One Second
7. FVC – Forced Vital Capacity
8. DALYs – Disability Adjusted Life Years
9. WHO – World Health Organization
10. NHLBI – National Heart, Lung And Blood Institute
11. CDC – Center For Disease Control
12. IJMR – Indian Journal Of Medical Research
13. CVA – Cerebro Vascular Accident
14. PR – Prevalence Rate
15. OR – Odds Ratio
16. CI – Confidential Interval
17. OPD – Out Patient Department
18. CxR – Chest X-Ray
19. URI – Upper Respiratory Tract Infection